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Wurfzahl und Wurffolge beim nordischen Wiesel (*Mustela nivalis rixosa* Bangs, 1896)

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Eingang des Ms. 12. 5. 1973

Bei ausreichendem Beutetierbestand sind beim mitteleuropäischen Mauswiesel (*Mustela nivalis vulgaris* Erxleben, 1777) zwei Jahreswürfe die Regel. Den Wieseln nördlicher Breiten steht infolge des erheblich kürzeren Sommers keine gleichlange Fortpflanzungszeit zur Verfügung. Dennoch ist ihre Vermehrungsfrequenz offensichtlich nicht geringer und nur erklärbar, wenn gleichfalls zwei Jahreswürfe angenommen werden (O. Kalela 1960 in mündl. Diskussion).

1964 konnte ich ein schwedisches Wiesel-Weibchen in Zucht nehmen, das nach Größe, Gewicht und Färbung der zirkumpolaren Unterart *rixosa* angehörte (genaue Daten in einer weiteren Veröffentlichung). Dieses brachte 1965, 1966 und 1967 je drei Würfe und bewies damit, daß auch das nordische Wiesel physiologisch auf mehr als einen Jahreswurf eingestellt ist. Daß es im natürlichen Verbreitungsgebiet mehr als zwei sein könnten, ist allerdings unwahrscheinlich. Der in Gefangenschaft produzierte dritte Wurf dürfte als Reaktion auf den längeren mitteleuropäischen Sommer

(die Zucht erfolgte in Oldenburg, 53°09'/8°13') zu werten sein und lediglich beweisen, daß das nordische Wiesel die zur Reproduktion geeignete Zeit in jedem Falle voll ausnutzt.

Bei dieser Gefangenschaftszucht zeigte sich weiter, daß die Tragzeit mit genau 5 Wochen der des mitteleuropäischen Mauswiesels entspricht und daß auch die Aufzucht der Jungen etwa die gleiche Zeit in Anspruch nimmt (erstes Beuteschlagen der Jungen ziemlich regelmäßig im Alter von knapp 6 Wochen, volle Unabhängigkeit von der Mutter mit 8—9 Wochen). Die Würfe folgten aber erheblich schneller aufeinander als bei den bis dahin in Zucht gehabten mitteleuropäischen Wiesel-Weibchen.

Dies resultierte daraus, daß das Schweden-Weibchen schon 5 Wochen (zweimal 34, zweimal 35, einmal 36 und einmal 38 Tage) post partum wieder brünstig wurde, was zweifelsfrei an seinem Verhalten erkennbar war: während es das Männchen bis dahin und noch am Tage davor mit Drohrufen abwies, begegnete es ihm nach Eintritt der Brunst mit anhaltendem Girren (Begrüßungs- und Lockruf) und ließ sich sofort zur Kopula ergreifen, die in allen sechs Testfällen zur Trächtigkeit führte. Da dies mit absoluter Regelhaftigkeit geschah, ist nicht daran zu zweifeln, daß eine physiologische Gesetzmäßigkeit vorlag und daß es sich nicht um ein individuelles, sondern um ein gruppenspezifisches Phänomen handelt.

Dieses hat zur Folge, daß der größere Teil der Tragzeit bereits während der Aufzucht des vorhergehenden Wurfes absolviert werden kann. Diese „Schachtelung" der Würfe ist möglich, weil die Jungen bereits vom 14. Lebenstage an von der von der Mutter zugetragenen Beute fressen, so daß die Milchproduktion rasch reduziert werden kann und das Muttertier während der folgenden Schwangerschaft kaum mehr belastet (Stillegung des ersten Zitzenpaares 3 Wochen, des zweiten 5 Wochen, des dritten 6 Wochen nach der Geburt der Jungen, so daß nur das vierte Paar bis zu deren Selbständigwerden in Funktion bleibt). Insgesamt werden damit zur Aufzucht von zwei Jahreswürfen nur $5^1/_2$ Monate benötigt.

Zweifellos sind die bei Carnivoren sonst wohl nicht vorkommenden „Schachtelwürfe" als Adaption an den kurzen nordischen Sommer zu werten. Es ist anzunehmen, daß diese „verkürzte" Wurffolge im gesamten zirkumpolaren Verbreitungsgebiet der systematisch offensichtlich einheitlichen nordischen Wiesel üblich ist. Ob auch die Mauswiesel südlicherer Breiten ihre Würfe „schachteln" können, ist ungewiß. Die *vulgaris*-Weibchen meiner Zucht ließen sich frühestens 9 bis 10 Wochen post partum, d. h. erst nach dem völligen Selbständigwerden des ersten Wurfes, wieder decken und benötigten für die Aufzucht ihrer zwei Jahreswürfe somit mehr als 7 Monate. Ein dritter Jahreswurf kam nicht vor, obwohl die Tiere in temperierten Räumen gehalten und ständig mit lebender Beute versorgt wurden. Das Material reicht aber nicht aus, um die Befähigung des mitteleuropäischen Mauswiesels zu zeitsparenden „Schachtelwürfen" definitiv auszuschließen und einen entsprechenden Unterschied in der Wurffolge mitteleuropäischer und nordischer Wiesel zu konstatieren, der angesicht der unterschiedlichen Sommerlänge im Verbreitungsgebiet beider Gruppen allerdings durchaus verständlich wäre.

Summary

Number and sequence of litters in the northern weasel

In each of three succeeding years, a nature-born captive female of the Scandinavian *Mustela nivalis rixosa* Bangs, 1896, produced three litters and thus confirmed the presumption that the weasels of northern latitudes are as capable of producing two litters per year as are those of temperate zones. Pregnancy (5 weeks) and rearing of the young (8—9 weeks) last the same time as in the Central-European *Mustela nivalis vulgaris* Erxleben, 1777. But the

mentioned female came in heat five weeks post partum so that the major time of the second pregnancy was already completed during the rearing of the first litter, and the rearing of two litters required about five and a half months. As this happened with punctual regularity, it is undoubtedly not an individual phenomenon but a general characteristic of northern weasels and to be interpreted as an adaption to the short summer of northern latitudes. Several captive females of the Central-European *M. n. vulgaris* did not come in heat until 9—10 weeks post partum, i. e. after the rearing of the young was fully completed. But the material is not sufficient for asserting their incapacity for a shortened litter sequence and for concluding a corresponding difference between the weasels of northern and temperate latitudes.

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Paradontal disease as a cause of tooth loss in a population of chamois *(Rupicapra rupicapra* L.) in New Zealand

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Receipt of Ms. 10. 9. 1973

Introduction

Paradontal disease causes the gradual destruction of tissues surrounding the tooth in the alveolus and the alveolar bone tissue, causing loss of one or more teeth. It has been reported in man (Coolidge and Hine 1958), domesticated (MacKinnon 1959; Cohrs 1954) and wild mammals (Eidmann 1939; Stubbe 1965; Rudge 1970 and Niethammer 1971).

The pathological course of paradontal disease is divided into three main phases (Coolidge and Hine 1958). The first phase — primary marginal gingivitis — begins when chemical or mechanical irritants cause the gingival tissue to become inflamed. The epithelium in the inflamed area becomes traumatic and open to bacterial infection. The second phase — periodontitis — occurs when the infection in the gingival tissue gradually penetrates through the gingival sulcus into the periodontal tissues, thereby affecting the periodontal membrane that anchors the tooth in the alveolus. This stage is accompanied by resorption of the alveolar bone crest; it is aggravated by accumulation and compaction of foreign material such as food, sand and grit within the lesion. The third phase — alveolar ostitis — begins when the infection penetrates deeper into the alveolus, causing destruction and resorption of alveolar bone. The principal connecting fibres of the periodontal membrane are affected and, because of unbalanced periodontal tension, teeth migrate from their normal position causing misalignment of the occlusal surface. With progression of the disease, as the alveolar bone itself becomes grossly infected, these teeth continue drifting until finally they are rejected. Healing of the infected area then takes place in those animals which have not succumbed to the disease. The external alveolar bone swelling is gradually resorbed, and the alveolus becomes filled with granular tissue which

Z. Säugetierkunde 39 (1974) 250—255

ISSN 0044—3468/ASTM-Coden ZSAEA 7

results in the expulsion of accumulated foreign matter (mainly compacted food). Finally, continuity of the oral mucuous membrane is restored over the now healed and filled alveolus.

This report describes the occurrence and incidence of paradontal disease in a population of chamois collected in New Zealand during the early southern summer of 1965–66.

Material and methods

Five hundred and seventy chamois right mandibles (lower jaws) of both sexes were collected for demographic analyses from the Copland and Rangitata river catchments in the South Island, New Zealand (CAUGHLEY 1970). The age of the male and female components was determined from growth rings in the cementum at the root of the first incisor (PKELHARING unpublished) and, when this tooth was absent, from growth restrictions on the horns (COUTURIER 1938).

Mandibles of mature animals were examined for symptoms of paradontal disease. Only the periodontitis, the alveolar ostitis and the final healed phase could be recognised. Mandibles arrived in the laboratory in a dried condition so that the marginal gingivitis phase could not be detected. In addition, as some of the incisors and canines were lost during transport, the study was restricted to molariform teeth.

Characteristics of the early and later periodontitis phase, the alveolar ostitis phase and the final healed phase were identified as follows:

1. Resorption of the alveolar bone crest (Fig. 1a).
2. Swelling of the mandible at the site of the infection (Fig. 1b).
3. Misalignment of teeth (Fig. 1c), usually accompanied by perforation of the jaw (Fig. 1d).
4. Missing teeth with the jaw still swollen (Fig. 1e).
5. Missing tooth or teeth with the alveolus filled and completely healed and no visible swelling of the mandible (Fig. 1f).

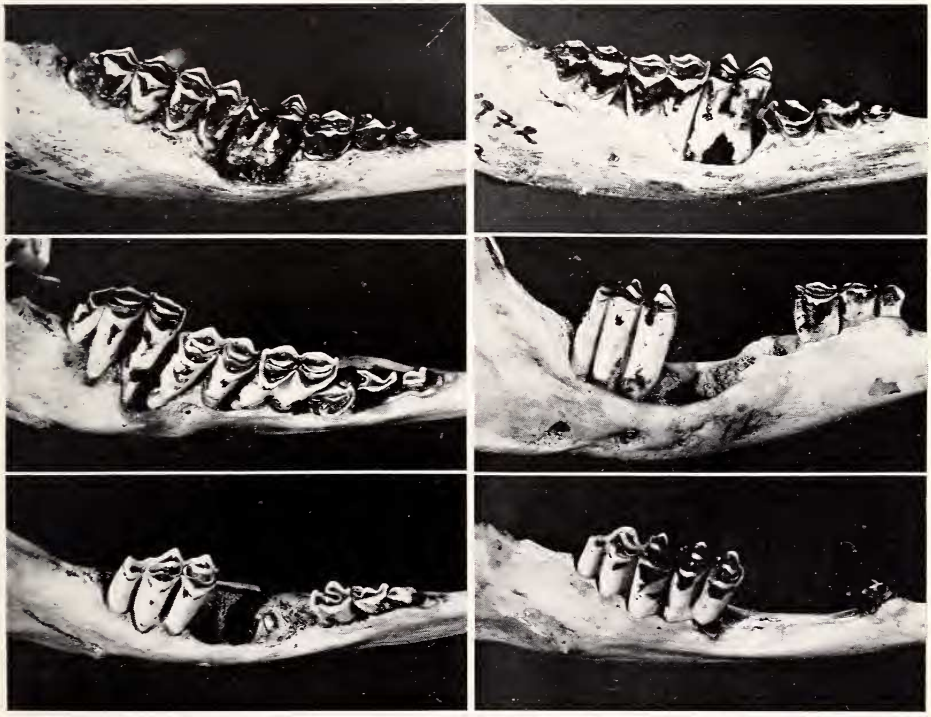

Fig. 1a—f. From left to right (Photographs by J. BURNIP)

Results

Of the 570 chamois of both sexes and all ages collected, 274 animals (183 ♀♀; 91 ♂♂) were older than 36 months of age and therefore had a completely erupted permanent set of molariform teeth (Couturier 1938). Mandibles of these animals were examined for symptoms of paradontal disease. Twenty per cent (N = 56) were affected by the later phases of paradontal disease (19 % of the females, n = 34; and 24 % of the males, n = 22). No significant difference ($P < 0.05$) was detected between the proportions of animals of each sex affected by paradontal disease (G-test, Sokal and Rohlf 1969).

To detect whether there was any difference in condition between diseased and healthy animals, males, pregnant and non-pregnant females were tested separately and age specifically by a two-factor, unbalanced analysis-of-variance. The two condition indicators tested were the kidney-fat-index (Riney 1955) and the weight of the kidney without the fat (Batcheler and Clarke 1970).

Despite the fact that the means of the diseased animals were consistently lower in all three categories tested, this difference was not great enough to outweigh the natural variance within each indicator. This may be because the age and sex specific sample sizes of diseased animals were inevitably small. The table shows the frequencies of healthy and diseased animals of both sexes in each age class. The difference between the proportion of healthy and diseased animals in each age cohort was highly significant for females ($X^2 = 14.5$; d. f. = 4; $P > 0.01$) and significant for males ($X^2 = 11.05$; d. f. = 4; $P > 0.05$). Therefore, it can be concluded that with increasing age, proportionally more animals were affected by paradontal disease. This suggests that the duration of the disease from the initial marginal gingivitis phase (not detected in this study) through to the later periodontitis and alveolar ostitis phases may be a drawn out process. It also suggests that susceptibility to infection may increase with age.

Age	Preg. + Non-pregnant ♀♀			♂♂		
	Diseased	Healthy	% Dis.	Diseased	Healthy	% Dis.
3	6	64	9	1	26	4
4	6	42	13	3	17	15
5	7	19	27	2	8	20
6	4	15	21	2	7	22
7+	7	9	44	8	11	42
	30	149		16	69	
	4 diseased animals not aged			6 diseased animals not aged		

Figure 2 shows the frequency for both sexes of infected alveoli retaining teeth and alveoli with rejected teeth. It appears that teeth with the longest occlusal stress, that is those that erupt first[1], show a higher frequency of infection with paradontal disease, even though they are not necessarily rejected in that order. This pattern was similar in both sexes. Molars are obviously not as easily rejected as premolars because of their longer and sturdier root system. McCall (1969) states that, in man, alveolar bone resorption caused by periodontitis will "first be seen around the teeth which erupt first; the first molar and the incisors". This is consistent with the above result.

[1] Eruption sequence of permanent lower molariform teeth in chamois; M_1 — M_2 — M_3 — [PM_2, PM_3, PM_4] (Couturier 1938).

The pathology of the disease in chamois is similar to the type description of periodontitis complex in man. In periodontitis simplex, bone is resorbed at an even rate around each tooth throughout the mouth and pockets are of even depth. In periodontitis complex, bone is resorbed unevenly in relation to individual teeth (Box 1921). Thus, pocket formation was localised and uneven in the alveolar tissues and around the infected tooth or teeth (see Fig. 1), and usually involved the first molar in both sexes. It was followed, in the early phases of alveolar ostitis by enlargement and deepening of the alveolus and accumulation of foreign matter. In the final phase loss of the tooth occurred.

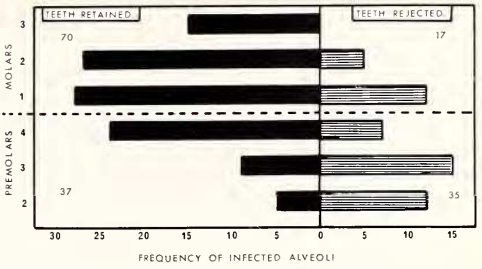

Fig. 2

Discussion

The frequency of animals infected by paradontal disease reported in this study is almost certainly an underestimate. This is because the primary marginal gingivitis phase could not be detected in the jaws of this sample; furthermore the study was restricted to molariform teeth in the lower right-hand jaws only. In several of these jaws molars were observed with abnormally elongated cusps, indicating an obvious gap in the maxillary toothrow.

There was no significant difference between the condition indices of diseased and healthy chamois in the sample studied. However, the extent of the damage caused to the alveoli of molars during the periodontitis and alveolar ostitis phases (see Figs. 1a, b, c and d) strongly suggests that animals affected by these later phases of paradontal disease were in the process of losing condition or would have lost condition. It is reasonable to assume that irritation during mastication causes reduced ingestion with a consequent lowering of the general condition. This assumption is supported by evidence given by MacKinnon (1959), and other reports published by Thompson (1906), Anon (1944), Murie (1944), Colyer (1947) and Coolidge and Hine (1958). However, the fact that some animals did survive the disease, although with a reduced dentition (see Fig. 1f) indicates that infection with paradontal disease is not necessarily fatal.

MacKinnon (1959) found no evidence in sheep of any generalised or other systemic disease (particularly of bone), and only the changes normally associated with falling condition and reduced intake of food. He also reported that in other cases of paradontal disease in sheep, he frequently observed the final healing phase after premolars had been rejected, but in the case of molars the animals usually had died or had been culled in an emaciated state before the repair process was complete. Rudge (1970) states however that in a population of feral goats on Macauley Island, Kermadec group, "the impact (of paradontal disease) on survival is not known but it probably was very slight, although it would certainly have been painful and affect chewing ability as age advanced".

Niethammer (1971) examined a sample of chamois skulls from New Zealand and concluded that both sexes show a greater dental variability than their ancestral Styrian counterparts. However, despite the fact that he observed symptoms associated with the later stages of paradontal disease, he did not consider that some of this